IN THE CLAIMS:

Amend the claims as follows:

Claims 1-15 (Cancelled).

16. (Currently Amended) An isolated nucleic acid comprising a nucleotide sequence encoding the effector domain of the binding molecule as claimed in claim 32~~claim 1~~.

17. (Currently Amended) An isolated nucleic acid comprising ~~as claimed in claim 16 wherein the~~ a nucleotide sequence ~~encodes~~ encoding a binding molecule as claimed in claim 32~~claim 1~~.

18. (Withdrawn) A nucleic acid as claimed in claim 16 which is a replicable vector.

19. (Withdrawn) A nucleic acid as claimed in claim 18 wherein the nucleotide sequence is operably linked to a promoter.

20. (Withdrawn) A host cell comprising or transformed with the vector of claim 19.

21. (Currently Amended) A process for producing a binding molecule as claimed in claim 32~~claim 1~~, the process comprising the step of modifying a nucleotide sequence

encoding a first human immunoglobulin heavy chain C_H2 such that 2, 3 or 4 amino acids in at least 1 region of the C_H2 domain corresponds to an amino acid from a second human immunoglobulin heavy chain C_H2 domain,

wherein the region is selected from the 2 discrete regions numbered residues 233-236, and 327-331 in accordance with the EU numbering system,

and wherein in each case the human immunoglobulin is selected from IgG1, IgG2 and IgG4.

22. (Withdrawn) A process as claimed in claim 21 wherein 2 amino acids in 1 region of the C_H2 domain are modified to the corresponding amino acids from a second human immunoglobulin heavy chain C_H2 domain.

23. (Currently Amended) A method of binding a target molecule comprising contacting said target molecule with ~~Use of~~ a binding molecule ~~or nucleic acid as claimed in claim 1~~of claim 32 under conditions allowing binding ~~to bind a target molecule with said binding molecule~~.

24. (Currently Amended) A method of ~~Use as claimed in~~ claim 23 wherein the target molecule is FcγRIIb, which binding causes inhibition of one or more of: B cell activation; mast cell degranulation; phagocytosis.

25. (Currently Amended) A method of ~~Use as claimed in~~ claim 24 to prevent, inhibit, or otherwise interfere with the binding of a second binding molecule to the target molecule.

26. (Currently Amended) A method of ~~Use as claimed in~~ claim 25 wherein the second binding molecule is an antibody.

27. (Currently Amended) A method of ~~Use as claimed in~~ claim 25 wherein the target molecule is selected from: the RhD antigen of red blood cells; an HPA alloantigen of platelets; a neutrophil antigen; a T-cell receptor; integrin; GBM collagen; Der P1; HPA-1a; VAP-1; laminin; lutheran; platelet glycoprotein VI; platelet glycoprotein Ia/IIa.

28. (Currently Amended) A method of ~~Use as claimed in~~ claim 24 for the treatment of a patient for a disorder selected from: Graft-vs-host disease; host-vs-graft disease; organ transplant rejection; bone-marrow transplant rejection; autoimmunity such as vasculitis, autoimmune haemolytic anaemia, autoimmune thrombocytopenia and arthritis; alloimmunity such as foetal/neonatal alloimmune thrombocytopenia; asthma and allergy; chronic or acute inflammatory diseases such as Chrohn's; HDN; Goodpastures, sickle cell anaemia, coronary artery occlusion.

29. (Currently Amended) A method of ~~Use as claimed~~ claim 23 wherein the binding molecule is administered to a patient, or optionally in cases where the patient is an unborn infant, to the mother of the patient.

Claim 30 (Canceled).

31. (Withdrawn) An oligonucleotide selected from:

MO22BACK: 5' TCT CCA ACA AAG GCC TCC CGT CCT CCA TCG AGA AAA 3'

MO22: 5' TTT TCT CGA TGG AGG ACG GGA GGC CTT TGT TGG AGA 3'

MO7BACK: 5' TCC TCA GCA CCT CCA GTC GCG GGG GGA CCG TCA GTC 3'

MO21: 5' GAC TGA CGG TCC CGC GAC TGG AGG TGC TGA GGA 3'

32. (Currently Amended) A binding molecule which is a recombinant polypeptide comprising:

(i) a binding domain capable of binding a target molecule, which binding domain is the binding site of an antibody, and

(ii) an effector domain having an amino acid sequence ~~substantially~~ homologous to all or part of a constant domain of a human immunoglobulin heavy chain;

wherein the binding molecule is capable of binding the target molecule without triggering significant complement dependent lysis, or cell mediated destruction of the target, and capable of specifically binding ~~FcRn and\or FcγRIIb~~ FcγRIIb and optionally FcRn,

and wherein the effector domain is a chimeric domain which is derived from two or more human immunoglobulin heavy chain CH2 domains, which human immunoglobulins are selected from IgG1, IgG2 and IgG4,

and wherein the effector domain has a reduced affinity for FcγRI, FcγRIIa and FcγRIII and a reduced ability to mediate complement lysis by comparison with said human immunoglobulin heavy chain C_H2 domains

and wherein the chimeric domain is a human immunoglobulin heavy chain CH2 domain which has the following blocks of amino acids at the stated positions: 233P, 234V, 235A and 236G and 327G, 330S and 331S numbered with respect to the EU numbering system of Kabat, and is at least 98% identical to a C_H2 sequence (residues 231-340) from human IgG1 or IgG4 having said modified amino acids.

33. (Currently Amended) The binding molecule as claimed in claim 32 wherein the effector domain is selected from G1Δac (SEQ ID NO:3) or G4Δc (SEQ ID NO:12) as shown in Figure 17.

Claim 34. (Cancelled)

35. (Previously Presented) The binding molecule as claimed in claim 32 wherein the effector domain is derived from a first human immunoglobulin heavy chain CH2 domain wherein at least 1 amino acid in at least 1 region of the CH2 domain has been modified to the corresponding amino acid from a second, different, human immunoglobulin heavy chain CH2 domain, and

wherein the effector domain has a reduced affinity for FcγRI, FcγRIIa or FcγRIII and a reduced ability to mediate complement lysis by comparison with the first or second human immunoglobulin heavy chain CH2 domain.

Claim 36. (Cancelled)

37. (Previously Presented) The binding molecule as claimed in claim 32 wherein the binding domain derives from a different source to the effector domain.

38. (Previously Presented) The binding molecule as claimed in claim 32 wherein the binding domain is capable of binding any of: the RhD antigen of red blood cells; an HPA alloantigen of platelets; a neutrophil antigen; a T-cell receptor; integrin; GBM collagen; Der P1; HPA-1a; VAP-1; laminin; lutheran; platelet glycoprotein VI; platelet glyprotein Ia/IIa.

39. (Currently Amended) ~~A binding~~ The binding molecule as claimed in claim 38 wherein the binding domain is selected from that of anti-CD52 antigen found on human lymphocytes; FOG1; OKT3; B2 (anti-HPA-1a); VAP-1; murine anti-α3 (IV) NC1; YTH12.5 (CD3); 2C7 (anti-Der p1); anti-laminin; or anti-lutheran.

40. (Previously Presented) A pharmaceutical preparation comprising a binding molecule as claimed in claim 32 plus a pharmaceutically acceptable carrier.

41. (Currently Amended) A binding molecule which is a recombinant polypeptide comprising:

(i) a binding domain capable of binding a target molecule, which binding domain is the binding site of an antibody, and

(ii) an effector domain having an amino acid sequence ~~substantially~~ homologous to all or part of a constant domain of a human immunoglobulin heavy chain;

wherein the binding molecule is capable of binding the target molecule without triggering significant complement dependent lysis, or cell mediated destruction of the target, and capable of specifically binding ~~FcRn and\or FcγRIIb~~ FcγRIIb and optionally FcRn,

and wherein the effector domain is a chimeric domain which is derived from two or more human immunoglobulin heavy chain CH2 domains, which human immunoglobulins are selected from IgG1, IgG2 and IgG4,

and wherein the effector domain has a reduced affinity for FcγRI, FcγRIIa and FcγRIII and a reduced ability to mediate complement lysis by comparison with said human immunoglobulin heavy chain C_H2 domains

and wherein the chimeric domain is a human immunoglobulin heavy chain CH2 domain which has the following blocks of amino acids at the stated positions: 233P, 234V, 235A and no residue at 236; and 327G, 330S and 331S, numbered with respect to the EU system of Kabat and is at least 98% identical to a CH2 sequence (residues 231-340) from human IgG1 or IgG2 having said modified amino acids.

42. (Previously Presented) The binding molecule as claimed in claim 41 wherein the effector domain is selected from G1Δab or G2Δa.

Claim 43. (Cancelled)

44. (Previously Presented) The binding molecule as claimed in claim 41

wherein the effector domain is derived from a first human immunoglobulin heavy chain CH2 domain wherein at least 1 amino acid in at least 1 region of the CH2 domain has been modified to the corresponding amino acid from a second, different, human immunoglobulin heavy chain CH2 domain, and

wherein the effector domain has a reduced affinity for FcγRI, FcγRIIa or FcγRIII and a reduced ability to mediate complement lysis by comparison with the first or second human immunoglobulin heavy chain CH2 domain.

Claim 45. (Cancelled)

46. (Previously Presented) The binding molecule as claimed in claim 41 wherein the binding domain derives from a different source to the effector domain.

47. (Previously Presented) The binding molecule as claimed in claim 41 wherein the binding domain is capable of binding any of: the RhD antigen of red blood cells; an HPA alloantigen of platelets; a neutrophil antigen; a T-cell receptor; integrin;

GBM collagen; Der P1; HPA-1a; VAP-1; laminin; lutheran; platelet glycoprotein VI; platelet glyprotein Ia/IIa.

48. (Currently Amended) The binding molecule as claimed in claim 47 ~~claim 38~~ wherein the binding domain is selected from that of anti-CD52 antigen found on human lymphocytes; FOG1; OKT3; B2 (anti-HPA-1a); VAP-1; murine anti-α3 (IV) NC1; YTH12.5 (CD3); 2C7 (anti-Der p I); anti-laminin; anti-lutheran.

49. (Previously Presented) A pharmaceutical preparation comprising a binding molecule as claimed in claim 41 plus a pharmaceutically acceptable carrier.